Emotion Awareness Predicts Body Mass Index Percentile Trajectories in Youth

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Objective To examine the rate of change in body mass index (BMI) percentile across 3 years in relation to emotion identification ability and brain-based reactivity in emotional processing regions.

Study design A longitudinal sample of 202 youths completed 3 functional magnetic resonance imaging–based facial processing tasks and behavioral emotion differentiation tasks. We examined the rate of change in the youth's BMI percentile as a function of reactivity in emotional processing brain regions and behavioral emotion identification tasks using multilevel modeling.

Results Lower correct identification of both happiness and sadness measured behaviorally predicted increases in BMI percentile across development, whereas higher correct identification of both happiness and sadness predicted decreases in BMI percentile, while controlling for children's pubertal status, sex, ethnicity, IQ score, exposure to antipsychotic medication, family income-to-needs ratio, and externalizing, internalizing, and depressive symptoms. Greater neural activation in emotional reactivity regions to sad faces also predicted increases in BMI percentile during development, also controlling for the aforementioned covariates.

Conclusion Our findings provide longitudinal developmental data demonstrating links between both emotion identification ability and greater neural reactivity in emotional processing regions with trajectories of BMI percentiles across childhood. *(J Pediatr 2015; ■: ■-■).*

Obesity plagues our nation's youths, with 31.7% of youths classified as overweight or obese.[1] Obesity during childhood and adolescence represents a significant pathway toward premature mortality and physical morbidity in adulthood.[2] As such, a better understanding of the psychological and related neurobiological correlates that may contribute to elevated body mass index (BMI) in youth is needed.

Emotion dysregulation,[3] the inability to effectively recognize and control intense emotional responses, has been hypothesized to contribute to the development of obesity in youth. Impaired emotional awareness, a component of emotion dysregulation, may be evidenced by an inability to correctly label and identify emotions in oneself and others, leading to distressing and emotionally dysregulated psychological states. Emotional reactivity, another component of emotion dysregulation, may be evidenced by increased activation in emotion-related brain regions, such as the amygdala. Excessive or impulsive eating may become a maladaptive mechanism through which children relieve their distress or attempt to self-regulate, leading to lack of attention to satiety[4] and weight gain. Decreased emotional awareness, still another component of emotion dysregulation, has been related to obesity in youth and adults[5-7] In a sample of Italian youth, a decreased ability to label others' emotions was associated with obesity status.[5] In a sample of female adolescents, the relationship between increased self-reported negative affect and overeating was mediated by poor emotional awareness.[4] Previous work has demonstrated a reduced ability to label visual and verbal signs of emotion in film clips in both obese children and their mothers.[6] Obese women also have been shown to exhibit deficits in emotional awareness and to more often report using eating as a strategy to regulate negative emotions compared with nonobese women.[7]

The foregoing findings suggest that obesity is related to difficulty in accurately identifying facial expressions of emotion; however, there is no published evidence linking poor emotional awareness to changes in BMI or obesity across time. The aforementioned studies support the hypothesis that poor emotional awareness would predict increases in BMI across time, eventually leading to obesity.

Another component of emotion dysregulation, emotional reactivity, has yet to be studied as a potential contributor to obesity in childhood. Emotional reactivity, which may present as an intense experience of negative emotions, could

BMI	Body mass index
BOLD	Blood oxygen level dependent
CAPA	Child and Adolescent Psychiatric Assessment
fMRI	Functional magnetic resonance imaging
MLM	Multilevel model
PAPA	Preschool Age Psychiatric Assessment
PDS	Preschool Depression Study

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lead to maladaptive regulation strategies, such as increased or loss of control eating. Functional magnetic resonance imaging (fMRI) studies in this area have primarily investigated reactivity in reward circuitry to images of food.[8] Findings from a recent meta-analysis indicate increased reactivity to food images in areas associated with explicit memory, such as the right parahippocampal gyrus, and reduced reactivity in regions linked to cognitive control, such as the dorsolateral prefrontal cortex, in individuals who are overweight or obese.[9]

To date, there have been few attempts to connect emotional reactivity to nonfood-related imagery to obesity or overweight status in childhood, however. In one study, overweight and obese girls with loss of control eating demonstrated a failure to engage brain regions associated with emotion regulation in response to a peer social rejection task.[10] Furthermore, heightened activation in the fusiform face area (associated with emotional processing) in overweight and obese girls with loss of control eating following a simulated peer rejection predicted subsequent food intake.

Given these preliminary findings of heightened activation to socially salient stimuli, investigating additional neural responses to faces (a different type of socially salient stimuli) in overweight and obese children is particularly important. Previous work has documented that brain regions involved in emotional reactivity, such as the amygdala, appear to be critical for the detection and processing of emotional faces.[11] Alterations in activity in these brain areas among youth at risk for becoming obese may suggest unique emotional processing characteristics related to high BMI.

In the present study, we investigated the relationship between neural reactivity to emotional faces during an emotional face processing task and BMI percentile trajectory in youths. We also examined the relationships between behaviorally based emotion identification abilities and the trajectory of BMI percentiles across childhood and early adolescence. We hypothesized that youths with poor emotion identification ability and greater neural activation to emotional faces in brain regions within an emotional reactivity brain network would evidence steeper increases in BMI across time, given that both factors are hypothesized to be indicators of greater emotion dysregulation. In this context, increased eating may serve as a maladaptive method of regulating the intense experiences of negative emotion (eg, emotional reactivity) and/or decreased ability to label and process emotions (eg, emotion recognition).

Methods

Participants were drawn from the Preschool Depression Study (PDS), a prospective longitudinal investigation of preschoolers and their families conducted at the Washington University School of Medicine.[12] The present study reports on 202 children from the PDS (n = 305 at baseline) who, across a 7-year period, completed annual behavioral assessments and at least 1 (out of a possible 3) fMRI scans proximal to these annual assessments. Parental written consent and child assent were obtained before participation, and the Washington University School of Medicine's Institutional Review Board approved all procedures.

Children who did not complete an fMRI scan reported more externalizing and internalizing symptoms compared with those who did complete an fMRI scan. There were no between-group differences in terms of BMI percentile, sex, puberty, ethnicity, IQ, family income-to-needs ratio, and major depressive disorder (MDD) symptoms.

Details of recruitment have been reported previously.[12,13] In brief, between 2003 and 2005, 3- to 6-year-old children were recruited from primary care practices and preschools/daycare centers throughout the St Louis metropolitan region using a screening checklist to oversample preschoolers with symptoms of depression. The final sample consisted of preschoolers with varying levels of depressive symptoms and healthy control preschoolers without psychiatric symptoms. In 2010, additional children (aged 10-15 years) with no previous psychiatric diagnosis were recruited from local schools to enhance the sample of healthy subjects. Children with neurologic or chronic medical problems or those with significant developmental delays were excluded. All PDS participants without contraindication for fMRI were eligible for an fMRI scan session. In the present study, 157 children (77%) were participants in the original PDS and 45 (23%) were recruited as additional healthy subjects in 2010.

Measures

Mothers reported family income at each annual assessment. The income-to-needs ratio was computed as the total family income at baseline divided by the federal poverty level, based on family size, at the time of data collection.[14]

The Kaufman Brief Intelligence test[15] was used to assess verbal and nonverbal intelligence at the behavioral assessment wave closest to scan 2. This test has a mean score of 100 (SD $\pm$ 15), and has proven to be a reliable and valid measure of IQ in youths.[16]

Exposure to medications was ascertained at each behavioral assessment closest to each scan using the MacArthur Health and Behavior Questionnaire.[17] The child's lifetime exposure to antipsychotic medications was deemed particularly important, given the known association between these medications and weight gain.[18] For the purpose of the present study, exposure to medication was coded as 0 for "no" and 1 for "yes."

Dimensional scores for externalizing (including attention deficit disorder, conduct disorder, and oppositional defiant disorder), internalizing (including generalized anxiety disorder, separation anxiety disorder, and posttraumatic stress disorder), and depressive symptoms were calculated at each behavioral assessment closest to each scan using the Preschool Age Psychiatric Assessment (PAPA)[19,20] and the Child

and Adolescent Psychiatric Assessment (CAPA).[21,22] Dimensional scores were created by summing all symptoms endorsed in each category. The PAPA and CAPA each consists of a series of developmentally appropriate questions covering the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* criteria for disorders of childhood. During the PAPA and/or CAPA interview, parents and children also identified their ethnicity. For the purpose of the present study, ethnicity was coded as 0 for white and 1 for African American or biracial.

The children's pubertal development was assessed at each behavioral assessment closest to each scan using the Pubertal Development Scale,[23] a self-reported instrument measuring various aspects of pubertal development on a 5-point Likert scale.

BMI Percentile

Trained research assistants recorded the children's height and weight (measured without shoes) at each behavioral assessment closest to each scan using a standardized procedure and a calibrated medical scale. BMI was calculated according to Centers for Disease Control and Prevention growth charts[24] using the standard formula (weight in kilograms divided by height in meters squared, rounded to 1 decimal place). Age- and sex-adjusted BMI percentiles were created using the formula and syntax available on the Centers for Disease Control and Prevention website.[24]

Emotion Identification

The 40-item version of the Penn Emotion Differentiation Test[25,26] was administered at each behavioral assessment closest to each fMRI scanning session to measure emotion identification of happy and sad faces. Each child viewed, via a computerized self-paced program, 40 pairs of faces, 1 pair at a time, and decided which face expressed the given emotion more intensely. This task has been shown to be reliable and valid in youths.[27] This variable was operationalized as the raw value of correct identification, with higher scores indicating more correct identification.

Neural Activation to Emotional Faces

Functional Task and Stimuli. PDS participants completed at least 1 scanning session (range, 1-3) that included task-based functional activation to emotional faces,[28] using an event-related facial emotion processing task similar to that used in previous research.[29-31] This report focuses on the sad and neutral faces, drawn from the MacArthur Network Face Stimuli Set[32] viewed by each child. This task did not explicitly focus children's attention on the emotional content of each face; rather, the children were simply asked to decide whether each face was male or female. Each sad expression was presented from 10 individuals at full and half intensity (full expression morphed with neutral),[28,33] with the primary focus on brain response to sad vs neutral faces (average functional activation to both the 100% and 50% intensity faces). The fMRI data were analyzed using in-house software (FIDL analysis package; http://www.nil.wustl.edu/~fidl).[34]

We obtained each subject's blood oxygen level dependent (BOLD) response to sad faces using fixed-effects general linear models incorporating regressors for linear trend and baseline shifts. A hemodynamic response shape was assumed (SPM canonical function) and used to derive magnitude estimates relative to fixation baseline. Each scan run consisted of 45 stimuli, 5 from each of the 9 conditions. Each stimulus was presented for 2500 ms, followed by an intertrial interval ranging from 500 to 6500 ms.[28]

fMRI Data Acquisition. Structural and functional scanning was performed on a 3.0-T MAGNETOM Trio Tim whole-body fMRI system (Siemens, Erlangen, Germany). BOLD images were acquired during face processing with a T2*-weighted asymmetric spin-echo echoplanar sequence (repetition time = 3000 ms, echo time = 27 ms, flip angle = 90°, field of view = 256 mm, voxel size = 4 × 4 × 4 mm) in the axial plane paralleling to the anterior-posterior commissure, with a 12-channel head coil. During each functional run, 99 sets of 36 contiguous axial images with isotropic voxels (4 mm^3) were acquired parallel to the anterior-posterior commissure plane.

fMRI Data Processing. fMRI data were preprocessed following standard steps, including: (1) compensation for slice-dependent time shifts; (2) removal of first 5 images of each run to allow the BOLD signal to reach steady state; (3) elimination of odd/even slice intensity differences owing to interpolated acquisition; (4) realignment of data acquired in each subject within and across runs to compensate for rigid body motion; (5) intensity normalization to a whole-brain mode value of 1000; (6) registration of the three-dimensional structural volume (T1) to the atlas representative template in the Talairach coordinate system[35] using a 12-parameter affine transform and resampling to 1-mm cubic representation[36,37]; (7) coregistration of the three-dimensional fMRI volume to the T2 and of the T2 to the participant's structural image; (8) transformation of the fMRI volumes to atlas space using a single affine 12-parameter transform; and (9) spatial smoothing using a 6-mm full-width half-maximum Gaussian filter. The common atlas template was optimized for children in our cohort's age range; the use of coregistration to this type of common template space has been validated in several previous studies.[38,39]

Previously validated "motion scrubbing" procedures adapted for fMRI data were used to remove artifacts resulting from head motion.[40,41] As discussed and validated previously for use in this sample,[42] the motion-scrubbing procedure assesses framewise displacement based on the parameters used in preprocessing step 4. This represents the differential head motion from one acquisition frame to the next summing across linear (x, y, z) and rotational displacements (yaw, pitch, and roll, where degrees of rotation are converted to millimeters of movement by calculating the displacement on the surface of a sphere with a

radius of 50 mm). Any frame with a sum displacement >0.9 mm was masked out of the analysis.

A Priori Region of Interest Definitions. As a measure of activation to emotional faces, we examined responses to sad faces vs neutral faces in a previously identified network of brain regions[43,44] associated with emotional responsivity. This network of regions included the bilateral amygdala and other regions that show positive resting-state functional connectivity with the amygdala, including hippocampal, parahippocampal, striatal, and temporal regions (emotional reactivity network; **Table I**, available at www.jpeds.com). For each region, we created a spherical region of interest, 12 mm in diameter, around its centroid (**Table I**). Magnitude estimates of the response to sad faces vs neutral faces were averaged across all of the regions of interest within the emotional reactivity network to create an overall estimate of responses in those regions.

Statistical Analyses

Analyses were conducted using SPSS version 21 (IBM, Armonk, New York). Repeated data on all covariates, main predictors (eg, emotion identification ability), and the outcome variable (BMI percentile) were available; therefore, we analyzed changes in BMI percentile across time (ie, using all 3 scan assessments) using multilevel models (MLMs)[45] to account for dependencies owing to repeated measurements. Each MLM examined the capability for happiness identification, sadness identification, or activation in emotional reactivity brain regions to sadness and happiness to serve as predictors (ie, each was assessed at 3 time points) of both the main effect and slope (ie, rate of change) in BMI percentile across time. Fixed effects were included in each MLM for time (ie, within-individual change over time in BMI percentile across the 3 scan assessments), correct happiness identification, correct sadness identification, emotional reactivity to sadness and happiness, and the interactions between time and emotion identification or emotional reactivity to examine the influence of these factors on changes in BMI percentile over time. All models included puberty, sex, ethnicity, IQ score, exposure to antipsychotic medication, family income-to-needs ratio, externalizing dimensional symptoms, internalizing dimensional symptoms, and depressive symptoms as covariates.

Before conducting analyses with the predictors of interest (eg, emotion identification and activation in brain regions), we ran a model that included only the covariates above to determine which exerted significant influence on BMI percentiles. None of the covariates had a significant effect on BMI percentile; however, given the theoretical and empirical support for both puberty and ethnicity being associated with differences in the rates of increase in BMI/BMI percentile, we chose to include interactions between these predictors and time in our final model. All continuous predictors were mean-centered. Random effects for the BMI percentile intercept and slope for time were included to account for individual variability in mean levels of BMI percentile and rate of change in BMI percentile.

Results

Descriptive statistics for all variables at each scan are presented in **Table II** (available at www.jpeds.com). At scan 1, participants ranged in age from 7 to 13 years. 108 participants were white and 94 participants were African American or biracial.

Performance-Based Measures of Emotion Identification of Faces in Relation to BMI

We examined correct happiness and sadness identification as predictors of the level and change in BMI percentile across time in 2 separate MLMs, controlling for the aforementioned covariates. We also conducted MLMs removing children with extremely low (eg, ≤3rd percentile) or extremely high (eg, ≥99th percentile) BMI and with age as a covariate instead of pubertal status. Substantive interpretations of the estimates of interest (all P values <.05) and model fit did not change (**Tables III** and **IV**; available at www.jpeds.com); therefore, to maintain statistical power, we chose to retain participants with extremely low or high BMI percentiles in the final analyses. We also chose to retain puberty in our analyses, given that puberty has been shown to be more closely associated with increases in BMI[46] and brain alterations[47] than age. There were main effects for correct happiness and sadness identification, indicating an association with average BMI percentile at scan 1 (**Table V**; available at www.jpeds.com). These main effects were qualified by interactions between emotion identification and time in predicting BMI percentile. The negative coefficient associated with each interaction term indicates that the changes in BMI percentile over time tends to be more strongly negative for individuals with more correct happiness and sadness identification. In addition, these interactions suggest that youths who had lower correct identification of either happiness or sadness showed increases in BMI percentile across time (**Figure 1**, A and B).

To aid in the interpretation of this effect, we tested the significance of the simple slopes at 1 SD below the mean, at mean, and at 1 SD above the mean of happiness and sadness identification. For happiness identification, the simple slope was 1.16 at −1 SD (P = .56), −1.95 at the mean (P = .35), and −5.05 at +1 SD (P = .06). Thus, as happiness identification ability increased, the slope of BMI across time became more strongly negative. The region of significance for happiness identification ranged from −12.69 to 4.79; approximately 14% of our sample had values of happiness identification (centered) falling outside of this range, indicating a simple slope that is significantly different from 0. This finding suggests that the rate of decrease in BMI percentile would be significant for relatively high levels of correct happiness identification.

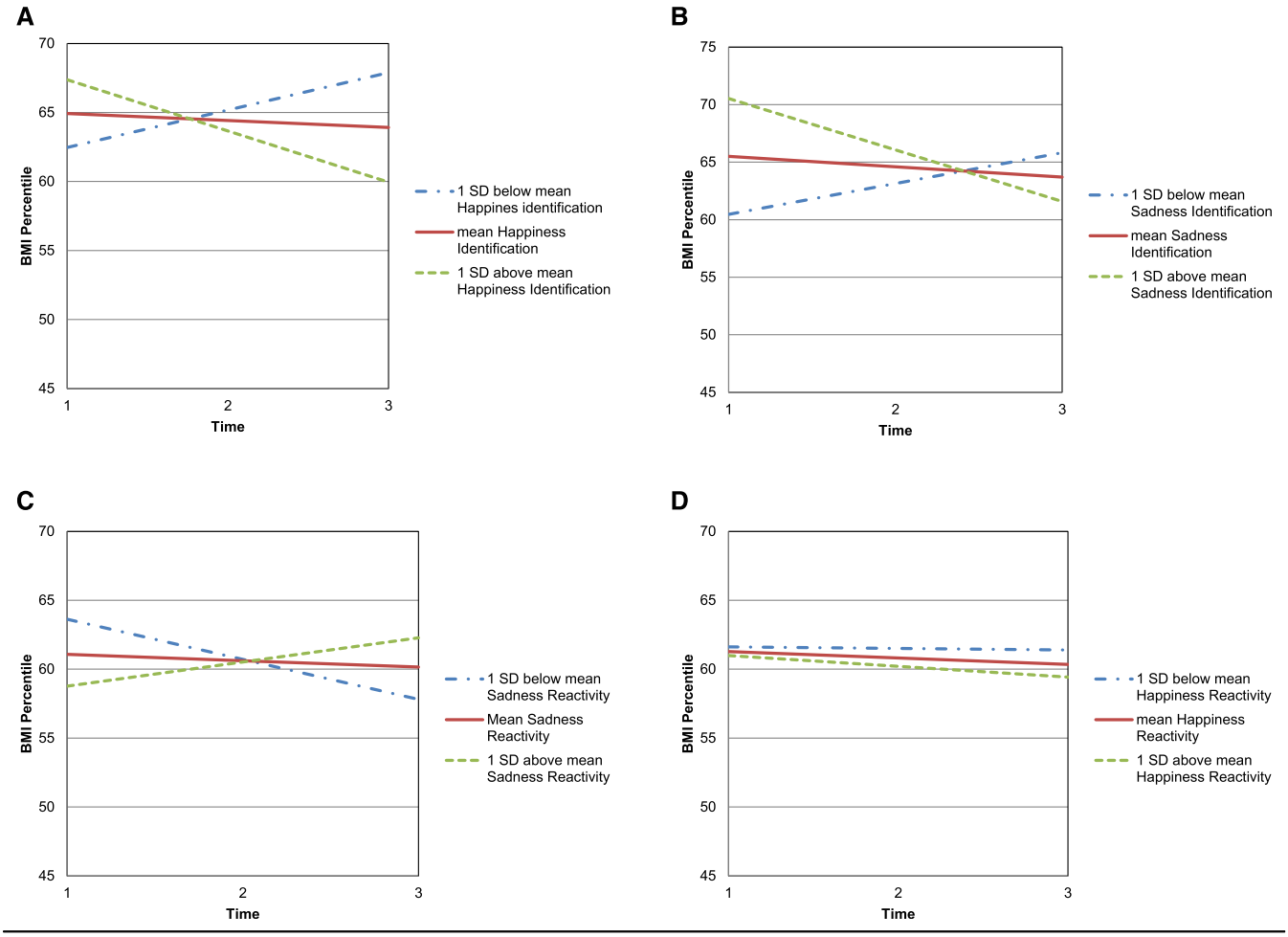

Figure 1. Model-implied trajectories of BMI percentiles at **A,** low, medium, and high levels of correct happiness identification; **B,** low, medium, and high levels of correct sadness identification; **C,** low, medium, and high levels of activation to sadness in emotional reactivity regions; and **D,** low, medium, and high levels of activation to happiness in emotional reactivity regions across time. Time indicates the scan number, such that 1 refers to each child's first scan and 3 refers to each child's third scan.

For sadness identification, the simple slope was 1.43 at −1 SD ($P = .51$), −2.04 at the mean ($P = .31$), and −5.51 at +1 SD ($P = .03$). Thus, as sadness identification ability increased, the slope of BMI across time became more strongly negative. The region of significance for sadness identification ranged from −7.26 to 2.22; Approximately 34% of our sample had values of sadness identification (centered) falling outside of this range, indicating a simple slope that is significantly different from 0. This suggests that the rate of decrease in BMI percentile would be significant for relatively high levels of correct sadness identification.

Neural Activation in Emotional Reactivity Regions to Sad and Happy Faces

We next examined functional brain activation in emotional reactivity regions to sad and happy faces as predictors of the level and change in BMI percentile across time, again controlling for the same variables above (Table V). We found no significant effects for reactivity to happiness on BMI percentile (Figure 1, D). There was a significant main effect for sadness reactivity on BMI percentile, indicating an association with BMI at scan 1. This main effect was qualified by an interaction between sadness reactivity and time. The negative coefficient associated with each interaction term indicates that the changes in BMI percentile over time tended to be more strongly negative for individuals with less reactivity to sad faces. In addition, this interaction suggests that youths with greater brain activation in emotional reactivity regions to sad faces had increases in BMI percentile over time (Figure 1, C).

To aid the interpretation of this effect, we tested the significance of the simple slopes at 1 SD below the mean, mean, and 1 SD above the mean of reactivity to sadness. The simple slope was −1.34 at −1 SD ($P = .47$), 1.03 at the mean of reactivity to sadness ($P = .50$), and 3.16 at +1 SD ($P = .08$). Thus, as reactivity to sadness increases, the slope of BMI across time becomes more strongly positive. The region of significance for reactivity to sadness ranges from −1.52 to 0.27;

approximately 8% of our sample had values of sadness reactivity (centered) falling outside of this range, indicating a simple slope that is significantly different from 0. This suggests that the rate of increase in BMI percentile would be significant for relatively high levels of sadness reactivity.

Exploratory Analyses

Based on BMI percentile at scan 1, we grouped children as healthy, designated 0 (eg, BMI percentile ≤84.99), or overweight/obese, designated 1 (eg, BMI percentile ≥85), at the time of scan 1. To determine whether these findings were driven by healthy or overweight/obese children, we ran our MLMs separately for each group. Group differences emerged for sadness identification, such that the interaction between sadness identification and time was significant only for children in the overweight/obese group at scan 1 (B = −1.55; $t = -2.84$; $P < .01$). Among children who were overweight/obese at scan 1, those who were able to accurately identify sad faces showed a decline in BMI percentile across time, whereas those who were worse at identifying sad faces showed an increase in BMI percentile across time (Figure 2).

To aid the interpretation of this effect, we tested the significance of the simple slopes at 1 SD below the mean, at the mean, and at 1 SD above the mean of sadness identification among overweight/obese children. The simple slope was 0.74 at −1 SD ($P = .79$), −3.69 at the mean ($P = .07$), and −8.12 at +1 SD ($P < .001$). Thus, as sadness identification ability increased, the slope of BMI across time became more strongly negative. The region of significance for sadness identification ranged from −11.29 to 0.19; approximately 30% of the obese children in our sample had values of sadness identification (centered) falling outside of this range, indicating a simple slope that is significantly different from 0. This finding suggests that the rate of decrease in BMI percentile would be significantly faster in children with high levels of correct sadness identification.

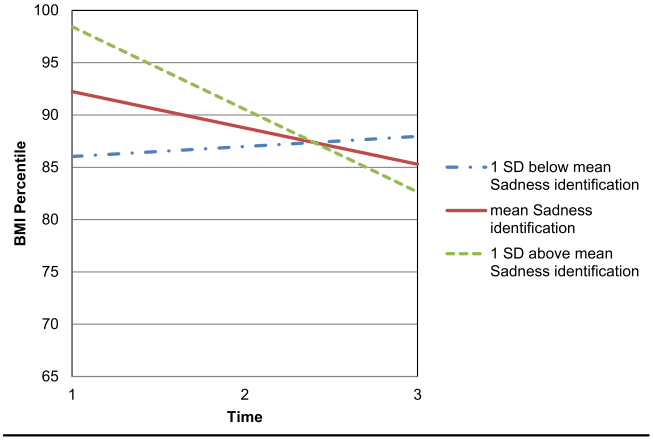

Figure 2. Model-implied trajectories of BMI percentiles at low, medium, and high levels of correct sadness identification across time among overweight/obese youth.

Discussion

This study used both neural measures of emotional reactivity and performance-based measures of emotion identification to predict changes in the developmental trajectories of BMI percentiles in youths. Our findings suggest that decreased ability to accurately recognize happiness and sadness in others and increased reactivity to sad faces are related to increases in BMI percentile throughout childhood. Conversely, increased ability to accurately recognize happiness and sadness in others and decreased reactivity to sad faces are related to decreases in BMI percentile throughout childhood. Among the children who were overweight or obese, an increased ability to recognize sad faces also led to a faster rate of decline in BMI across time. These findings emerged even when dimensional measures of psychopathology and other key factors that could contribute to BMI percentiles were accounted for in the analyses. Such findings provide data that could meaningfully inform intervention efforts aimed at stabilizing the rate of increase or even enhancing the rate of decrease in the trajectories of BMI in youths on a pathway toward obesity.

We found that in children, a poor ability to correctly identify sad and happy faces predicted increases in BMI percentile across time and that an above-average ability to correctly identify sad and happy faces predicted decreases in BMI percentile across time, while controlling for symptoms of psychopathology and relevant demographic variables. Of note, the significance of these interactions occurred primarily at the extremes of happiness and sadness identification (eg, very high and low values), indicating that for most youths with average emotion identification ability, there is no significant association with BMI percentile changes across time. This finding in a sample of youths with varying BMI is consistent with what has been reported in obese children and adults who show reduced awareness of their own and others' emotions,[6,7,48] and adds to the growing body of literature linking social-emotional processing deficits with eating disorders.[49,50] Our present findings advance our understanding of the dynamic interplay between social-emotional development and physical well being by demonstrating that poor facial emotion recognition ability may be one pathway toward accelerated weight gain across childhood, possibly contributing to obesity in adolescence or early adulthood. Furthermore, these findings offer an alternative approach to weight management or loss in children at risk for developing obesity: focusing on the accurate recognition of emotions.

Additional fMRI results indicate that greater neural activation to sad faces, but not to happy faces, in brain regions within the emotional reactivity brain network predicted increases in BMI percentile across time, whereas less neural activation to sad faces in the same brain regions predicted decreases in BMI percentile across time, again controlling for clinical and other relevant variables. The significance of this

interaction also occurred at the extreme values of sadness reactivity, and there was no significant correlation between BMI percentile change and sadness reactivity for youths evidencing average sadness reactivity. Differences in the brain function of youths with more rapidly increasing BMI percentile trajectories, as evidenced by greater neural responses to sad faces in the emotional reactivity network, may indicate difficulty with regulation of negative emotion arising from top-down brain regions thought to modulate activation of emotional reactivity regions. Previous work has indicated greater reactivity to food-related imagery among obese individuals,[8] but this is one of the first studies to link BMI trajectories across development in youths to neural responses to nonfood-related emotional stimuli. Thus, obese youths and those at risk for obesity as a function of rapidly increasing BMI may exhibit deficits in emotion awareness, particularly sadness, not restricted to food.

Taken together, our findings suggest that reduced emotion identification ability, particularly within the context of increased neural reactivity in the emotional processing brain regions, may place youths at risk for increasing BMI. Youths may be experiencing heightened brain-based reactivity to emotional faces and at the same time be unable to accurately label facial affect, a combination that places an individual at high risk for distress and impairment. Furthermore, results from this study suggest that among youths who are already overweight or obese, those who exhibit an increased ability to identify facial emotions show a faster rate of decline in BMI percentile over time. Additional longitudinal research linking responses in these brain regions to emotion identification performance and BMI throughout childhood that accounts for other key psychosocial, genetic, and metabolic risk factors, such as physical activity and family history of obesity, is warranted.

This study could not assess more broad family-level factors that have been shown to be associated with childhood obesity, such as parental obesity. Despite this limitation, it is unlikely that such family-level factors would entirely account for the relationships found in this study. Although evidence exists for substantial heritability in both obesity[51] and facial affect processing,[52] environmental factors also make significant contributions. It is crucial that future work continue to explore the unique and shared contributions of family- and child-level factors that influence changes in BMI over time. Genetic contributions to BMI were not measured in this study, and additional work is needed on the genetic contributions to obesity and how family- and child-level factors may influence heritability. Although the number of participants in this study is large for neuroimaging work, the sample is relatively small for assessing obesity. In addition, the study sample contains a high proportion of children with psychopathology; however, this was accounted for in the analysis. Furthermore, given the relatively small number of children with an extremely low BMI percentile, these data cannot inform whether similar patterns may be exhibited in youths at risk for other eating pathology, such as anorexia.

This study significantly adds to the limited research on developmental trajectories of emotion awareness, emotional reactivity, and BMI percentile in youths using a racially and socioeconomically diverse sample. One strength of this study is the use of 2 objective emotion paradigms that were repeated across time, one that is behavioral/performance-based and another that captures neural reactivity to sad faces through fMRI. Consistent findings were shown for both tasks, indicating that both emotion recognition and greater neural responses to faces in emotional reactivity brain regions were related to changes in BMI percentile over time.

Taken together, these findings have implications for the treatment and/or prevention of obesity in youth. Given that youths experienced increases in BMI percentile when correct emotion identification ability was low, interventions targeting this aspect of emotional competence may be a particularly useful for youths who are overweight or obese. Further supporting such an approach, youths who were overweight or obese actually showed the fastest rate of decline in BMI percentile when their performance on these tasks was high. The ability to accurately label emotions in others may increase awareness of one's own emotions, lessening physiological feelings of distress. Over time, enhanced emotion awareness may decrease episodes of emotionally driven or uncontrolled eating and, as a result, stop significant weight gain.[4] ■

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Table I. Regions included in the emotional reactivity network

	Region		
Emotional reactivity network	**x**	**y**	**z**
Left amygdala	−23	−8	−16
Left hippocampus	−27	−26	−12
Left inferior temporal gyrus	−41	−9	−29
Left superior temporal gyrus	−27	6	−32
Right amygdala	18	−8	−20
Right hippocampus	34	−26	−14
Right parahippocampal gyrus	18	−23	−14
Right putamen	25	2	−7
Right superior temporal gyrus	33	10	−28

Table II. Descriptive statistics at each scan

	Scan 1 (n = 172)			Scan 2 (n = 154)			Scan 3 (n = 150)		
	Mean	**SD**	**Range**	**Mean**	**SD**	**Range**	**Mean**	**SD**	**Range**
Age, y	10.28	1.27	7-13	11.74	1.20	9-15	12.86	1.16	10-16
Female sex, %	49			49			49		
Puberty	1.74	0.98	1-4	2.43	1.02	1-4	2.99	0.96	1-5
White ethnicity, %	52			52			52		
Externalizing dimensional symptoms	4.59	6.03	0-29	3.58	4.89	0-24	2.90	4.83	0-29
Internalizing dimensional symptoms	2.29	2.62	0-17	1.71	1.87	0-13	1.38	1.62	0-10
MDD dimensional symptoms	2.54	2.13	0-9	1.89	1.71	0-8	1.94	1.62	0-6
Antipsychotic medication use, %	7			7			7		
Correct happiness identification	8.96	4.26	2-17	9.23	3.77	2-18	10.07	3.55	2-18
Correct sadness identification	10.63	3.64	3-18	10.80	2.80	4-17	11.43	2.56	5-16
BMI	19.29	4.53	12-38	20.04	4.66	13-41	21.26	5.19	14-43
BMI percentile	60.10	31.23	1.47-99.59	59.56	31.71	1.03-99.62	62.44	30.79	1.29-99.63

MDD, major depressive disorder.
Puberty coded as 1, prepubertal; 2, early puberty; 3, mid-pubertal; 4, late puberty; 5, postpubertal. Sex coded as 1, male; 0, female. Ethnicity coded as 0, white; 1, African American. Happiness and sadness identification indicates the number of correct trials.

Table III. Fixed-effects estimates from the MLMs predicting change in BMI percentile from emotion identification and emotional reactivity with extremely low and high BMI percentiles removed

Effects	B	SE	*t*	df	*P*
Happiness					
Intercept	77.97	9.76	7.98	301.29	.00
Scan	−2.37	2.13	−1.11	169.06	.27
Sex	0.28	4.59	0.06	182.23	.95
Puberty	−1.78	2.85	−0.62	172.09	.53
Ethnicity	−21.00	7.93	−2.65	227.14	.01
IQ	0.22	0.18	1.22	178.16	.23
Income-to-needs	0.36	2.05	0.17	310.56	.86
Externalizing dimensional symptoms	0.43	0.31	1.41	207.12	.16
Internalizing dimensional symptoms	−0.47	0.77	−0.62	186.92	.54
MDD symptoms	0.91	0.70	1.30	182.00	.19
Antipsychotic use	−5.09	8.25	−0.62	276.58	.54
Correct Happiness ID	1.42	0.81	1.75	165.30	.08
Puberty*Scan	1.23	1.21	1.02	156.46	.31
Ethnicity*scan	2.95	2.73	1.08	151.89	.28
Happiness ID*scan	−0.80	0.36	−2.25	157.92	.03
Sadness					
Intercept	77.74	9.62	8.08	299.35	.00
Scan	−2.35	2.07	−1.14	169.78	.26
Sex	0.35	4.62	0.08	182.53	.94
Puberty	−2.29	2.87	−0.80	170.18	.42
Ethnicity	−20.20	7.63	−2.65	227.23	.01
IQ	0.17	0.18	0.97	179.07	.33
Income-to-needs	0.37	2.05	0.18	311.55	.86
Externalizing dimensional symptoms	0.48	0.30	1.60	201.93	.11
Internalizing dimensional symptoms	−0.62	0.76	−0.82	183.44	.42
MDD symptoms	1.04	0.70	1.50	178.93	.14
Antipsychotic use	−4.63	8.26	−0.56	281.37	.57
Correct sadness ID	2.81	1.01	2.79	165.50	.01
Puberty*scan	1.34	1.22	1.09	154.91	.27
Ethnicity*scan	2.35	2.578	0.91	149.34	.36
Sadness ID*scan	−1.14	0.45	−2.52	159.12	.01
Emotional reactivity to sadness					
Intercept	65.01	8.63	7.53	263.32	.00
Scan	0.65	1.53	0.42	169.17	.67
Sex	2.43	4.51	0.54	184.10	.59
Puberty	0.27	2.10	0.13	179.68	.90
Ethnicity	−7.07	6.36	−1.11	184.56	.27
IQ	0.17	0.18	0.96	184.55	.34
Income-to-needs	−0.08	1.85	−0.04	366.65	.97
Externalizing dimensional symptoms	0.54	0.30	1.80	295.08	.07
Internalizing dimensional symptoms	−0.65	0.62	−1.04	240.82	.30
MDD symptoms	0.46	0.64	0.72	242.97	.47
Antipsychotic use	−1.21	7.56	−0.16	259.97	.87
Sadness reactivity	−27.46	12.51	−2.20	212.66	.03
Puberty*scan	0.33	0.92	0.36	181.05	.72
Ethnicity*scan	−3.13	1.87	−1.67	127.56	.09
Sadness reactivity*scan	13.46	6.34	2.13	209.60	.04
Emotional reactivity to happiness					
Intercept	66.63	8.62	7.73	262.19	.00
Scan	0.65	1.54	0.42	167.95	.67
Sex	2.59	4.48	0.58	183.89	.56
Puberty	0.35	2.12	0.17	180.31	.87
Ethnicity	−7.64	6.30	−1.21	186.09	.23
IQ	0.18	0.18	0.99	185.74	.32
Income-to-needs	−0.23	1.86	−0.12	366.50	.90
Externalizing dimensional symptoms	0.48	0.30	1.60	298.64	.11
Internalizing dimensional symptoms	−0.61	0.63	−0.96	243.46	.34
MDD symptoms	0.38	0.65	0.59	247.53	.56
Antipsychotic use	−3.10	7.55	−0.41	256.55	.68
Happiness reactivity	0.78	13.85	0.06	200.20	.96
Puberty*scan	0.30	0.93	0.33	182.20	.74
Ethnicity*scan	−2.86	1.88	−1.52	126.16	.13
Happiness reactivity*scan	−2.48	6.75	−0.37	194.89	.71

ID, identification; *MDD*, major depressive disorder.
Scan denotes scan number across time. Sex coded as 1, male; 0, female. Ethnicity coded as 0, white; 1, African American. Happiness and sadness identification indicates the number of correct trials.

Table IV. Fixed-effects estimates from the MLMs predicting change in BMI percentile from emotion identification and emotional reactivity with age as a covariate instead of puberty

Effects	B	SE	*t*	df	*P*
Happiness					
Intercept	78.25	9.80	7.99	298.65	.00
Scan	−2.01	2.69	−0.75	299.12	.46
Sex	2.56	4.53	0.56	181.39	.57
Age	−0.12	2.82	−0.04	209.47	.97
Ethnicity	−19.81	7.84	−2.53	235.50	.01
IQ	0.24	0.18	1.33	185.48	.19
Income-to-needs	0.48	2.03	0.24	317.11	.81
Externalizing dimensional symptoms	0.39	0.30	1.32	205.81	.19
Internalizing dimensional symptoms	−0.37	0.75	−0.50	190.88	.62
MDD symptoms	0.94	0.67	1.41	183.66	.16
Antipsychotic use	−7.04	8.03	−0.88	282.68	.38
Correct happiness ID	1.24	0.78	1.59	170.15	.11
Age*scan	0.44	0.97	0.45	152.88	.65
Ethnicity*scan	1.80	2.58	0.70	158.75	.49
Happiness ID*scan	−0.73	0.34	−2.17	164.75	.03
Sadness					
Intercept	78.93	9.68	8.15	296.49	.00
Scan	−2.20	2.68	−0.82	300.18	.41
Sex	2.48	4.55	0.55	181.17	.59
Age	−1.31	2.82	−0.46	207.65	.64
Ethnicity	−19.66	7.55	−2.60	234.21	.01
IQ	0.20	0.18	1.09	186.14	.28
Income-to-needs	0.64	2.03	0.32	318.36	.75
Externalizing dimensional symptoms	0.45	0.30	1.54	199.99	.13
Internalizing dimensional symptoms	−0.50	0.74	−0.67	186.81	.50
MDD symptoms	1.05	0.66	1.58	179.70	.12
Antipsychotic use	−7.00	8.02	−0.87	286.89	.38
Correct sadness ID	2.67	0.97	2.75	170.94	.01
Age*scan	0.86	0.99	0.87	152.83	.38
Ethnicity*scan	1.45	2.46	0.59	157.12	.56
Sadness ID*scan	−1.14	0.43	−2.64	166.71	.01
Emotional reactivity to sadness					
Intercept	65.28	9.00	7.26	267.16	.00
Scan	0.69	2.31	0.30	319.99	.77
Sex	4.05	4.50	0.90	183.93	.37
Age	1.36	1.95	0.70	265.40	.49
Ethnicity	−7.74	6.36	−1.22	191.84	.22
IQ	0.19	0.18	1.06	188.28	.29
Income-to-needs	−0.07	1.79	−0.04	369.19	.97
Externalizing dimensional symptoms	0.53	0.29	1.82	297.41	.07
Internalizing dimensional symptoms	−0.61	0.59	−1.03	244.36	.31
MDD symptoms	0.54	0.61	0.89	243.41	.38
Antipsychotic use	−1.49	7.59	−0.20	264.55	.84
Sadness reactivity	−26.25	12.00	−2.19	213.86	.03
Age*scan	−0.33	0.68	−0.48	190.33	.63
Ethnicity*scan	−3.48	1.80	−1.93	127.96	.06
Sadness reactivity*scan	12.83	6.11	2.10	211.06	.04
Emotional reactivity to happiness					
Intercept	67.13	8.99	7.47	266.31	.00
Scan	0.55	2.33	0.24	319.42	.81
Sex	4.18	4.48	0.93	184.25	.35
Age	1.30	1.96	0.66	262.27	.51
Ethnicity	−8.39	6.31	−1.33	193.29	.18
IQ	0.20	0.18	1.10	189.58	.27
Income-to-needs	−0.30	1.80	−0.17	370.76	.87
Externalizing dimensional symptoms	0.46	0.29	1.58	299.05	.12
Internalizing dimensional symptoms	−0.52	0.60	−0.87	246.40	.38
MDD symptoms	0.46	0.62	0.75	246.44	.45
Antipsychotic use	−3.40	7.59	−0.45	261.39	.65
Happiness reactivity	1.03	13.57	0.08	206.28	.94
Age*scan	−0.26	0.69	−0.38	191.49	.71
Ethnicity*scan	−3.15	1.82	−1.73	128.92	.09
Happiness reactivity*scan	−2.23	6.63	−0.34	202.29	.74

MDD, major depressive disorder; *ID*, identification.
Scan denotes scan number across time. Sex coded as 1, male; 0, female. Ethnicity coded as 0, white; 1, African American. Happiness and sadness identification indicates the number of correct trials.

Table V. Fixed-effects estimates from the MLMs predicting change in BMI percentile from emotion identification and emotional reactivity

Effects	B	SE	*t*	df	*P*
Happiness					
Intercept	76.80	9.72	7.90	305.15	<.001
Scan	−1.95	2.06	−0.94	172.73	NS
Sex	1.11	4.61	0.24	185.68	NS
Puberty	−2.35	2.79	−0.84	174.36	NS
Ethnicity	−21.25	7.92	−2.68	237.35	.01
IQ	0.23	0.18	1.29	183.13	NS
Income-to-needs	0.36	2.02	0.18	314.61	NS
Externalizing dimensional symptoms	0.42	0.30	1.40	210.06	NS
Internalizing dimensional symptoms	−0.29	0.75	−0.39	188.90	NS
MDD symptoms	0.80	0.68	1.17	183.86	NS
Antipsychotic use	−4.88	8.25	−0.59	283.50	NS
Correct happiness ID	1.42	0.79	1.80	168.48	.07
Puberty*scan	1.40	1.17	1.19	159.56	NS
Ethnicity*scan	2.63	2.67	0.99	156.71	NS
Happiness ID*scan	−0.82	0.34	−2.39	161.75	.02
Sadness					
Intercept	77.14	9.57	8.06	304.01	<.001
Scan	−2.04	2.00	−1.02	173.85	NS
Sex	1.22	4.63	0.26	186.96	NS
Puberty	−2.81	2.78	−1.01	172.48	NS
Ethnicity	−20.71	7.60	−2.72	237.72	.01
IQ	0.19	0.18	1.05	184.79	NS
Income-to-needs	0.47	2.02	0.23	316.83	NS
Externalizing dimensional symptoms	0.49	0.30	1.64	205.50	NS
Internalizing dimensional symptoms	−0.44	0.74	−0.60	185.69	NS
MDD symptoms	0.93	0.68	1.37	180.86	NS
Antipsychotic use	−4.71	8.25	−0.57	289.31	NS
Correct sadness ID	2.85	0.98	2.92	169.13	.00
Puberty*scan	1.51	1.17	1.29	158.47	NS
Ethnicity*scan	2.08	2.52	0.83	154.87	NS
Sadness ID*scan	−1.21	0.44	−2.78	162.27	.01
Emotional reactivity to sadness					
Intercept	64.89	8.68	7.48	268.44	<.001
Scan	0.91	1.52	0.60	173.31	NS
Sex	3.47	4.52	0.77	187.17	NS
Puberty	−0.10	2.07	−0.05	186.30	NS
Ethnicity	−7.96	6.38	−1.25	190.26	NS
IQ	0.19	0.18	1.05	187.19	NS
Income-to-needs	−0.08	1.80	−0.05	369.10	NS
Externalizing dimensional symptoms	0.53	0.30	1.80	299.26	.07
Internalizing dimensional symptoms	−0.57	0.60	−0.95	239.94	NS
MDD symptoms	0.46	0.63	0.73	246.68	NS
Antipsychotic use	−1.73	7.59	−0.23	264.09	NS
Sadness reactivity	−26.36	12.10	−2.18	213.89	.03
Puberty*scan	0.39	0.91	0.43	186.34	NS
Ethnicity*scan	−3.20	1.85	−1.74	129.69	.09
Sadness reactivity*scan	12.99	6.16	2.11	210.69	.04
Emotional reactivity to happiness					
Intercept	66.66	8.66	7.70	267.54	<.001
Scan	0.87	1.53	0.57	172.61	NS
Sex	3.59	4.50	0.80	187.05	NS
Puberty	−0.10	2.09	−0.05	186.21	NS
Ethnicity	−8.64	6.32	−1.37	191.80	NS
IQ	0.20	0.18	1.09	188.24	NS
Income-to-needs	−0.32	1.81	−0.17	370.25	NS
Externalizing dimensional symptoms	0.46	0.30	1.55	301.74	NS
Internalizing dimensional symptoms	−0.49	0.60	−0.81	242.00	NS
MDD symptoms	0.40	0.64	0.63	250.83	NS
Antipsychotic use	−3.68	7.59	−0.48	260.73	NS
Happiness reactivity	1.55	13.72	0.11	206.16	NS
Puberty*scan	0.38	0.92	0.41	187.30	NS
Ethnicity*scan	−2.87	1.86	−1.54	128.77	NS
Happiness reactivity*scan	−2.70	6.68	−0.40	200.51	NS

NS, not significant; *MDD*, major depressive disorder; *ID*, identification.
Scan denotes scan number across time. Puberty coded as 1, prepubertal; 2, early puberty; 3, mid-pubertal; 4, late puberty; 5, postpubertal. Sex coded as 1, male; 0, female. Ethnicity coded as 0, white; 1, African American. Happiness and sadness identification indicates the number of correct trials.